

# A Sticky Wicket: Opposing Functions of p120-Catenin in Development and Cancer

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It has been known for some time that cell-cell adhesion mediated by E-cadherin and catenins is important for development and cancer in epithelial tissues. Although  $\beta$ -catenin is upregulated in many cancers, p120-catenin is downregulated in most human cancers. Before this study, the molecular mechanism underlying  $\beta$ -catenin function in tumorigenesis was well on its way to being worked out, but little was known about p120. This paper piqued my interest because it showed that p120 was a major regulator of E-cadherin stability in the salivary gland. Its loss caused a major decrease in E-cadherin with severe defects in cell-cell adhesion and tissue morphology resembling intraepithelial hyperplasia. I liked this paper because it made me think about how looking at development gives important insights into cancer and about how two related molecules that bind to E-cadherin could behave in opposite ways to get the initial phases of tumorigenesis started.

This PaperPick refers to “Blocked Acinar Development, E-Cadherin Reduction, and Intraepithelial Neoplasia upon Ablation of p120-Catenin in the Mouse Salivary Gland,” by M.A. Davis and A.B. Reynolds, published in January 2006.